post-procedural period. Further study is warranted to investigate a strategy of administration of c7E3 *after* coronary dissection has occurred.

1017-69 Preconditioning Human Myocardium — Reduced Hypoxanthine Production During PTCA

Nigel Jepson, Kameljit Kalsi [1], Richard Cowell, Vincent Paul, Charles Ilsley, Anne-Marie Seymour [1]. *Dept. of Cardiology, NHLI, Harefield Hospital, Middlesex, U.K.; [1] Dept. of Cardiothoracic Surgery, NHLI, Harefield Hospital, Middlesex, U.K.*

Brief episodes of myocardial ischaemia and reperfusion protect against subsequent prolonged ischaemic insults. This adaptive response termed preconditioning has been observed with successive coronary occlusions at PTCA based on recordings of anginal intensity and ECG changes. The aim of this study was to evaluate myocardial release of Hypoxanthine (HX) (an ATP catabolite and a sensitive marker of ischaemia) in coronary sinus blood immediately after successive balloon inflations. Nine male patients mean age 50 ± 2.1 with isolated left anterior descending artery stenoses and normal left ventricular function were studied. Collateral channels filling diseased arterial segment were absent on the diagnostic angiogram. PTCA consisted of three balloon inflations, durations of which were: (1) 60–90 seconds, (2) 90–180 seconds, (3) 180–300 seconds with 5 minutes of intervening reperfusion between each. Paired aortic and coronary sinus blood samples were taken immediately after each deflation and deproteinized with 1.3 M $HClO_4$ and neutralized supernatant fractions analysed by HPLC for HX content. The arteriovenous differences (mean ± SEM) were:

	Before PTCA	Reperfusion		
		1	2	3
Hypoxanthine (μmoles/litre of blood)	−0.01 ± 0.37	−3.41 ± 2.05	−1.54 ± 0.64	−2.42 ± 0.56

Hypoxanthine release is reduced after second and third prolonged balloon inflations when compared to the first shorter inflation reflecting an adaptive response of the myocardium to ischaemia. These results show metabolic evidence for preconditioning in the human myocardium.

1017-70 A New Low Cost Protocol for Elective PTCA

Mark Nootens, Joseph Condon, Greg Elsner, Brenda Erner, Ronald Landin, Charles Orr, James Hermiller, Cass Pinkerton, Thomas Linnemeier. *The Indiana Heart Institute, Indianapolis, IN*

In order to curb costs, 2 protocols for elective PTCA were developed. Path "A" pts had sheath removal ASAP following PTCA (when ACT < 150) and discharge after 12 hrs IV heparin. Path "B" pts had overnight heparin with sheath removal the following morning. "A" pts had no venous sheath, type and screen, IV NTG or labs except PTT drawn post PTCA. "B" pts had a venous sheath, type and screen, IV NTG and labs drawn post PTCA (lytes, BUN/creat, CPKx3). Unstable angina, acute MI, staged PTCA or stents were excluded. "A" or "B" was used for elective PTCA, at the discretion of the angiographer, from 7/93–7/94 and compared to prior elective PTCA, 7/92–7/93 (prepath).

	Pts	LOS	Compl	Costs ($)			Savings
				Lab	Room	Total	
Prepath	819	2.1	1.6%	2,670	825	3,495	
Path "A"	115	1.04	0.9%	2,582	590	3,172	9.2%
Path "B"	227	1.28	0.9%	2,654	643	3,297	5.7%

LOS = length of stay (days), Compl = death, MI, CVA or hematoma requiring vascular repair or transfusion, Lab = cath lab, supplies and blood work costs, Room = PTCA unit and telemetry costs

Conclusion: In elective PTCA costs are reduced safely by discharge the next day (path "B") and further in selected pts by using a new low cost protocol (path "A"). Additional savings might be appreciated if these pts were observed in a less intense setting after sheath removal.

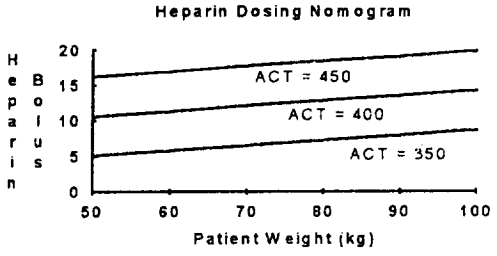
1017-71 Patient-Specific Heparin Dosing During Angioplasty

William B. Hillegass, Brigitta C. Brott, Eric B. Haura, Robert A. Harrington, Harry R. Phillips, Richard S. Stack, Robert M. Califf. *Duke Hospital, Durham, NC*

The risk of ischemic events and bleeding complications during angioplasty is related to the intensity of heparin anticoagulation as monitored by the activated clotting time (ACT). Data on patient characteristics that affect the dosing of heparin during angioplasty to achieve a desired target ACT is limited. The Hemochron ACT was measured 10 minutes after a heparin bolus in 438 non-emergency angioplasty patients. The influence of heparin dose, patient age, weight, gender, presenting diagnosis, and pre-procedural continuous intravenous heparin use on the intensity of anticoagulation achieved were examined with univariate followed by stepwise multivariate regression analysis.

	Univariate		Multivariate	
	critical value	p-value	critical value	p-value
Age	0.034	ns		
Weight	−2.15	0.033	−2.925	<0.001
Female Gender	1.21	0.34		
Unstable Angina	−2.04	0.043	−0.069	ns
Pre-procedural IV Heparin	−5.38	<0.001	−2.39	0.023
Heparin Dose	6.35	<0.001	8.95	<0.001

Conclusions: Patient weight, pre-procedural IV heparin therapy, and heparin dose were significant independent predictors of the ACT achieved. Pre-procedural intravenous heparin significantly increased the heparin dose required to achieve the target ACT, suggesting the development of tolerance to heparin. Similarly, patients with unstable angina required significantly more heparin to achieve the target ACT. Recognition of these factors allows patient-specific heparin dosing during angioplasty. A nomogram for patients receiving pre-procedural heparin therapy, for example, shows the relationship between patient weight, heparin dose, and the expected target ACT.



1017-72 Correlates of Acute Myocardial Infarction (AMI) After Successful Coronary Angioplasty (PTCA)

Rizwan A. Karatela, William S. Weintraub, John S. Douglas Jr, Spencer B. King III. *Emory University School of Medicine, Atlanta, GA*

To identify the factors predicting AMI after PTCA we studied 10,772 patients who had successful PTCA at our institution. 880 patients had AMI following discharge while 8,754 patients remained AMI free during long term follow up after successful uncomplicated PTCA. While there was no difference in the 5 year freedom from AMI based on sex, presence of hypertension or LVH, the freedom from AMI was lower in patients with previous MI (84% vs 91%, p = < 0.0001), diabetes (84% vs 90%, p = < 0.0001), angina class III–IV (87% vs 91%, p = < 0.0001), and CHF (85% vs 89%, p = 0.014). The multivariate correlates of AMI were:

	Chi SQ	ODDS Ratio	p Value
Previous MI	37	1.49	<0.0001
Angina Class III–IV	20	1.17	<0.0001
Diabetes	13	1.46	<0.0001
Multivessel CAD	11	1.26	0.001
Age	8	0.98	0.004
CHF	5	1.34	0.024

In long term follow up after successful angioplasty, the presence of these co-morbid illnesses may help in identifying the patients at higher risk of having AMI after successful angioplasty. This risk stratification strategy may influence survival after angioplasty.